First, the presence of tubercle bacilli, without which, of course, tuberculosis could not arise; secondly, dust in the air; and, thirdly, gaseous impurities. Dust in the air, especially minute fragments of mineral matter, by damaging the epithelium of the air-passages, allows a point of entrance to the bacilli, and gaseous impurities, especially carbonic dioxide, by diminishing the protective power of the lymphatic glands, favour the growth of the bacilli. If time had permitted, I might have directed attention to the entrance of other infectious diseases via the upper respiratory tract, but I think that I have brought forward sufficient material to give rise to a useful discussion, especially as I am to be followed by Dr. Jobson Horne.

Introductory Remarks by W. Jobson Horne, M.D.,

Surgeon to the Metropolitan Ear, Nose, and Throat Hospital, Ernest Hart Memorial Research Scholar of the British Medical Association.

It is unnecessary to dwell upon the importance of the subject under discussion; its importance is rivalled only by the vastness of the field it covers, and this will become apparent as the discussion develops. An increasing recognition of the part played by the upper respiratory tract as a source of systemic infection must be attributed, I think, to the improved methods of clinical analysis in modern medicine, a natural concomitant of the growth of specialism.

It would be idle, and, after the exhaustive paper by Dr. de Havilland Hall, it is unnecessary, for me in the present communication to attempt to pass in review in the brief time at my disposal the vast range of diseases which can gain admittance to the body through the portals which fall within the domain of our practice. It would be equally idle for me to attempt to discuss any one of these diseases in all its amplitude.

All that I propose doing is to invite your attention to the consideration of a few diseases of obscure origin and insidious onset, which, I believe, more frequently than is commonly thought have their origin in the upper respiratory tract, and to the part played by the lymphatic system in modifying or arresting their progress. The diseases to which I propose alluding are: Infective endocarditis, tuberculosis, lymphadenoma (Hodgkin's disease), and lymphosarcoma.

The term "infective endocarditis" in its usually accepted and restricted sense, meaning the malignant, ulcerative, or septic phase of the disease, is perhaps, in the light of our present knowledge, no

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longer tenable, for we now know that acute endocarditis, inasmuch as it is associated with one or other of the infective diseases, is always infective in nature. The cases I am about to refer to, however, belong to the more malignant and destructive variety, in which the septic process is by no means confined to the heart, but the stress of the disease is upon that organ.

The part played by the faucial tonsils in rheumatic fever, and therefore in acute endocarditis, has already been submitted for your consideration, and I need not dwell upon that point.

I pass down to the larynx, a breach in the mucous membrane of which may be the site of entry of the parasite, which will bring about the destruction of the patient. Do not let it be thought for one moment that I am desirous of establishing the larynx to the exclusion of other sites of infection as the *fons et origo* of this disease, or of any of the other diseases to which I shall allude. I merely give you post-mortem-room facts and clinical observations which cannot be lightly passed over.

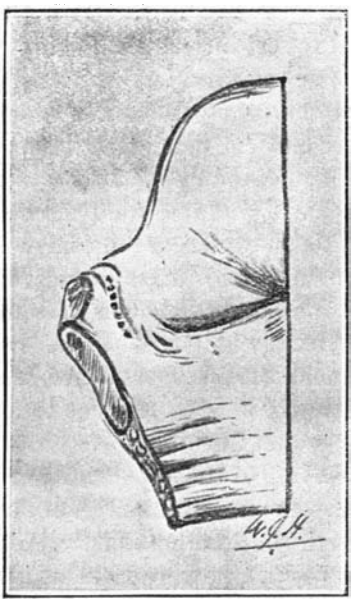
It is now nearly ten years since I first observed a case of malignant endocarditis in which the primary infection was in the larynx. It occurred in a man aged fifty-seven. At the autopsy the cusps of the mitral and aortic valves presented vegetations and ulcerations, the larynx in its left half was swollen and sloughy, and the left arytenoid cartilage was necrosed and exfoliating. The lesions in the larynx were so gross as to leave no doubt about it being the primary site of infection.

More recently I have had the opportunity of observing a similar case, the larynx from which is in the museum at St. Bartholomew's Hospital; and through the kindness of Dr. Andrewes, the curator, I am able to show you a photograph in which it will be observed that both arytenoid cartilages are necrosed and exfoliated. The laryngeal lesions in these two cases were so advanced that it was difficult to indicate the precise site of infection. I have, however, a larynx from another case—a younger subject—of malignant endocarditis, in which death occurred at an earlier stage, and which shows the starting-point of the ulceration. This point is in a fold of mucous membrane, commencing behind the vocal process, and passing obliquely upwards and backwards, to be lost between the summits of the cartilages of Santorini and Wrisberg. I find this to be a site which lends itself to infection in other diseases about which I shall speak; here secretion may rest and stagnate, and here stress and strain is put upon the laryngeal mucosa. I shall refer to it as the vulnerable spot.

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The upper respiratory tract as a source of tuberculous infection may be considered under three headings:

Firstly, it may be the site of direct infection. I use the term in contradistinction from indirect infection, which I shall almost immediately explain. A tuberculous lesion in this region may be the outcome of direct infection by the virus from outside the body, as, for example's sake, primary tuberculosis of the nose, the fauces, the post-nasal adenoid tissue, the soft palate, the pharynx, the parotid gland, the tongue, the larynx, and the ear. All of such primary infections, with the exception of the last, are comparatively rare. On the other hand, the lesions may be the outcome, and



A Diagram of the Interior of the Left Half of a Larynx to show the Site referred to as the Vulnerable Spot, which is indicated by a Dotted Line.

this is more commonly the case, of direct inoculation with sputum from the lungs, in which case, of course, the infection is a secondary one; and in illustration of this I would instance secondary tuberculosis of the larynx and the ear. Primary tuberculosis of the larynx is so extremely rare that it is a negligible quantity. The diagnosis of such a condition, for obvious reasons, can only be based upon post-mortem evidence. In the course of ten years, whilst closely following the post-mortem examinations of a large general hospital, I have never met with a single case.

From dead-house observations, I feel that I am now in a position to make the following statements with reference to tuberculosis of the larynx:

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1. When the larynx is infected with tubercle, the disease is already established in the lungs.

2. That by the time the disease in the larynx has advanced to ulceration, the disease in the lung has advanced to cavitation.

3. When the disease in the lung is confined to the pure miliary form, the larynx is never infected.

These statements, it will be noted, are uttered with a degree of dogmatism to which I am seldom given. The deductions to be drawn from them is obvious—that infection of the larynx takes place by the sputum. The bearing of these observations upon the so-called cure of laryngeal tuberculosis by means of surgical treatment must be equally obvious and undeserving of comment.

Infection of the ear by the sputum I have dwelt upon so recently in a discussion at the Otological Society of the United Kingdom that I think I need not detain you on that point.

I would refer here to a matter of controversy which closely concerns our discussion—namely, the relative potency of the virus of tuberculosis in its dry and moist forms. Without entering into any discussion about the propagation of tuberculosis, I think we may assume it is generally agreed that the spread of the disease is to be attributed mainly to the particles of sputum from phthisical patients. Cornet and his school hold that the sputum is most dangerous when it has become dry and pulverized. Flügge maintains that the sputum is more infectious in the moist form, as in the spray produced in coughing and sneezing. Numerous experiments have been done by these two schools in support of their respective views. Let us consider what light is thrown upon the subject by the references I have made in connection with tuberculosis of the ear and larynx. Both of these regions are exposed to infection through the agency of an atmosphere charged with tuberculous dust, yet it is extremely rare to meet with tuberculosis primary in the larynx by direct infection from the air inspired. It is equally rare to meet with tuberculosis in the ear starting from the external meatus. On the other hand, evidence has been adduced to prove that infection by the sputum direct from its mother soil to the larynx and to the orifice of the Eustachian tube is by no means rare. These facts seem to me to strongly support the view advanced by Flügge.

Tuberculous ulceration, when it occurs in the trachea, is usually more marked on the posterior wall, and is met with in subjects of advanced phthisis who have become bedridden. Doubtless the prolonged contact of a tenacious agent such as sputum fully explains the etiology and situation of the lesions observed.

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Secondly, let me consider the upper respiratory tract as the path of indirect systemic infection with tuberculosis; by that I mean infection through the upper respiratory tract, without any naked-eye or positive macroscopic evidence of the path of infection being left behind.

Lymphoid tissue, of all tissues, is most vulnerable to tuberculosis, and yet how difficult it is to demonstrate tubercle bacilli in excised tonsils, post-nasal adenoids, or lymphatic glands. Were one to diligently microscope excised tonsils and adenoids, cutting a series of sections through the entire structure, the reward for one's pains would be to find giant-cell systems in about 4 per cent. and tubercle bacilli in rather less than 1 per cent. of such examinations, notwithstanding the fact that the material may have been taken from cases of tuberculous adenitis, and perhaps primary tuberculosis of the middle ear. This is an important pathological fact, to which I shall revert.

That a systemic infection with tuberculosis does take place through the lymphoid ring of the upper respiratory tract is beyond question. It was demonstrated by Sims Woodhead in his experiments on swine, which show that the lungs can become infected through the cervical glands by feeding with infected material. It is, of course, difficult to demonstrate the truth of this in the human subject.

The preceding considerations bring me to my next proposition —namely, the part played by the lymphoid tissues in modifying a systemic infection. Call to mind the cases of tuberculous adenitis in which the disease is restricted under hygienic measures to the cervical glands. Infection in such cases must have taken place through the upper respiratory tract. Couple with this clinical observation the pathological difficulty in demonstrating tubercle bacilli from the lymphoid tissues from these regions, and we are driven to consider whether, through some property of the tissue, the bacilli having set up a morbid process may perish or become crippled amidst the lesions they have engendered, and the systemic infection is thereby arrested or, at least, modified in its progress.

This brings me to the consideration of another disease—namely, lymphadenoma, or Hodgkin's disease. I prefer in the present state of our knowledge using the latter term. It is comprehensive and non-committal in sense. Far be it from me to provoke a discussion here as to whether lymphadenoma is or is not tuberculosis. At present I recognise in it a histological, but not a pathological, entity. As regards the etiology, it has long been surmised that Hodgkin's disease is due to an infection. When we bear in mind that, so far as it is possible to ascertain, Hodgkin's disease has commenced in the glands of the neck in at

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least 50 per cent. of the cases recorded, and also that the tonsils themselves are not infrequently affected in this disease, it is reasonable to point to the latter structure as a probable point of entrance of the infecting agent, whatever it may be. I have already published cases demonstrating ulceration of the larynx as a point of entrance of the infection,[1] and I think the evidence brought forward in those cases is ample to indicate the necessity of examining the larynx as a possible site of infection in the diseases grouped under Hodgkin's name.

Lastly, I wish to refer to a case of thoracic lymphosarcoma which occurred in a man aged forty-nine. At the autopsy it was found that the mediastinum contained a considerable mass of new growth, presenting the microscopic appearance of a lymphosarcoma. The main bronchus on the right side, although not invaded, was decidedly compressed, but not occluded by the growth; the œsophagus had been impinged upon to an extent of 4 inches, and for about 2 inches had been seriously narrowed. The gland at the bifurcation of the trachea was greatly enlarged, and appeared to be the starting-point of the growth. The anterior and posterior glands in the mediastinum were involved. The other glands in the body were not obviously affected.

The right lung near its root contained a secondary deposit of the circumference of a penny piece. No microscopic evidence of tubercle was met with in the lungs or in any other part of the body. The liver and spleen were free from new growth.

The larynx on the outer side of the right arytenoid region presented a circumscribed area of œdema about the size of a raisin. On the inner aspect of the right arytenoid there was the puckered scar of an ulcer, situated immediately below the cartilages of Santorini and Wrisberg, in the situation I have referred to as the vulnerable spot.

Under the microscope, the case was found to be one of sarcoma; that is to say, histological round-celled sarcoma was present both in the growth and in the secondary deposit in the lung. After all, "sarcoma" is but a mere term, and conveys no more precise information about the nature of the case than the term "lymphadenoma" would afford about that of the others I have referred to. The presence of the ulcer in the larynx in this particular case raises the interesting question whether the growths were not the result of an infection, and whether sarcoma may not eventually have to be numbered together with the lesions met with in Hodgkin's disease with the infective granulomata.

[1] Journal of Laryngology, Rhinology and Otology, December, 1901.

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Before concluding, I wish to thank our President for his address, eloquently appealing for a compulsory training of students in laryngoscopy. In my brief remarks I have only touched upon a very small part of a subject of vast importance in general medicine, and even that has been done imperfectly, but I trust what little I have said will be accepted as further evidence in support of our President's appeal. *Non cuivis homini contingit adire Corinthum.* To behold Corinth was to the civilized Roman the acme of culture. Let us hope that in the near future we shall cease to have cause to sing, *Non cuivis medico contingit adire laryngem.*

Discussion.

Dr. J. L. Goodale (Boston, Mass.) read a paper dealing chiefly with the histology of the tonsils in their relation to disease, of which the following is a summary:

The object of the following paper is to discuss certain chronic disturbances of the system arising through the absorption of bacterial products from the lymphoid tissue of the upper respiratory tract. We have here to do with conditions which are essentially different from those occurring in acute infections. In the latter class of cases the pathogenic micro-organism invades the tissues, either in virtue of its virulence, or as a result of the temporary lowering of the resistance of the organism. In the chronic conditions, on the other hand, alterations have occurred in the histological or anatomical structure of the tissues which permit the development on or in them of saprophytic bacteria, and the disturbances produced are due to the absorption into the system of products of decomposition.

In order to understand the etiology of these conditions, it will be necessary to bear in mind certain histological points relating to the structure of lymphoid tissue. I will remind you that the unit of lymphoid tissue is the follicle—a collection of lymphoid cells enclosing a central endothelial reticulum. These units are separated from each other by an endothelial network, containing lymphoid cells, plasma cells, and cells of a type intermediate between these two. The surface of this tissue is formed by mucous membrane, squamous or columnar, of considerable density, equal at least in compactness to the mucous membrane of the vicinity. In the pharyngeal, faucial, and lingual tonsils, the stout mucous membrane of the free surface forms invaginations, which are lined with comparatively loose mucous membrane. This is the important circumstance which renders these organs points of inferior resistance against the invasion of micro-organisms. Fluids and finely-divided

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particles in contact with the compact epithelium of the free surface of these tonsils penetrate with difficulty or not at all. When introduced into the crypts, they have been observed to penetrate the intercellular deficiencies of the lacunar epithelium, and travel with the efferent lymph stream between the follicles and towards the capsule of the organ, and even into the cervical lymph glands. The situation of these tonsils is thus seen to be an important factor in determining the method of resistance against bacterial invasion. The pharyngeal tonsil is situated in a region in contact with air which has already been filtered nearly or quite free of bacteria. The secretion of the numerous mucous glands of the vicinity acts as a protective covering to prevent the direct contact of micro-organisms against the mucous membrane. Finally, the invaginations of the mucous membrane present little or no opportunity for the retention and decomposition of substances in contact with them.

In the case of the lingual tonsil, the lacunæ are comparatively short and wide-mouthed, while a constant stream of fluid is passing by with each act of swallowing. Retention and decomposition of the lacunar contents is thus practically impossible.

In the faucial tonsils, on the other hand, a variety of factors come into play in determining this question. Under normal circumstances, the crypts are irrigated by saliva and other fluids with almost the same degree of thoroughness as in the case of the lingual tonsil. The leucocytes and cellular detritus of the crypts have thus no opportunity to accumulate. Under certain conditions, however, free egress from the lumen of the crypt is restricted, and decomposition of its contents ensues. The two chief factors in causing this condition are acute inflammation and retrograde metamorphosis.

As shown by the writer, acute tonsillitis is characterized histologically by a diffuse inflammation of the parenchyma of the organ, appearing in the form of an increased proliferation of the lymphoid cells and of the endothelial cells of the reticulum, due to the absorption of a toxin formed in the crypts. The crypts are filled with exfoliated epithelial cells, leucocytes, bacteria, amorphous débris, and, in some cases, fibrin. The leucocytes are chiefly polynuclear neutrophiles, many of which contain bacteria in their interior. Some show nuclear fragmentation, with dispersion of their chromatin. There are also seen smaller numbers of lymphoid cells, plasma cells, and cells intermediate in character between these two. Of chief importance in the present connection is the different degree of intensity of the process in the distal and

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proximal portions of the crypts. Bacteria are abundant near the orifice of the crypt, gradually diminishing towards the base, which at times seems nearly free from them. The phagocytic leucocytes correspondingly near the orifice of the crypt exhibit the greatest number of incorporated bacteria. In cases of especial clinical severity, fibrin is seen along the walls of the lacunæ near the orifice as a delicate network enclosing cells and bacteria. In the deeper portions of the crypt, fibrin occurs only in exceptional cases. We thus see that in acute tonsillitis the activity of the process is chiefly manifested near the distal portion of the crypt. It is thus evident that denudations of the epithelium and adhesions of the opposite walls would occur more readily near the orifice of the crypt than near its base. If the adhesions completely occlude the orifice of the crypt, a cystlike cavity is formed in the tonsil. If this cavity is free from bacteria, it will exert no further influence upon the system, except possibly through pressure. If, however, the occlusion is not complete, but permits the entrance of saprophytic bacteria, decomposition of the lacunar contents rapidly ensues.

Inflammatory adhesions of the tonsillar folds to the surface of the organ may bring about the same result by partially closing the orifice of one or more crypts.

With regard now to retrograde metamorphosis, as age advances contractions and alterations occur in the structure of the tonsil of great variety. The resulting condition depends upon the manner in which this sclerosis takes place. If the tonsil has been previously essentially normal, it merely retracts more or less symmetrically into its recess between the anterior and posterior pillars. If, on the other hand, the crypts have been previously dilated, and if the free surface is more or less covered by adhesions and folds of membrane extending to the vicinity, the process of involution will be attended by an irregular narrowing of the lumen of the crypts. If the narrowing begins near the base of the crypt, and extends from this point outward, no harm will result; but if the narrowing, on the other hand, begins at the orifice, a bottle-shaped condition of the crypt will be produced, with resulting faulty drainage and decomposition of its contents.

In the histological examination of tonsils which are the seat of putrefactive processes, we are struck first with the relatively large size of the endothelial centre of the follicles. This enlargement is out of proportion to the number of lymphoid and plasma cells in the vicinity, and is due to increased proliferation of the endothelial cells lining the reticulum. In response to irritation these endothelial cells give rise to cells having the appearance of epithelial

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cells, but with phagocytic properties. Their phagocytosis is not only for amorphous detritus, but also for other living cells, chiefly red blood corpuscles and lymphoid and plasma cells. It differs thus from the phagocytosis exhibited by the polynuclear neutrophiles of the crypts, which is limited to micro-organisms and detritus. It differs, however, from the enlargement found in acute inflammation in that there is no swelling of the cytoplasm of the endothelial cells of the bloodvessels, or abnormal exfoliation. The proliferation and phagocytosis is to be regarded as the result of the absorption into the interior of the tonsil of irritating toxines produced by the saprophytic bacteria of the crypts.

In several instances I have found large concealed terminal dilatations of the lacunæ. In these cases clinical examination showed a tonsil of moderate size, with apparently normal crypts. After excision, however, a cavity was encountered, adjacent to the capsule, containing a large amount of fœtid, puriform fluid. The existence of such an accumulation may be difficult to recognise clinically, even after careful probing or division of the intra-lacunar tissue.

These conditions of saprophytic development, whether apparent or concealed, are sufficient to bring about a definite train of clinical symptoms, which may be briefly referred to. Pallor, impairment of strength and spirits, fœtid odour of the breath, gastric disturbances of various kinds, are most frequently found. Abnormal susceptibility to acute infections is frequent. In two instances I have seen obstinate acne of the face, which had been treated by skilled dermatologists for a number of years, with but slight transitory improvement, show a complete clearing up of the skin after a deep-seated, concealed, fœtid collection had been removed from the tonsil.

Dr. Frederick Poynton said: Dr. de Havilland Hall's able introduction of this important subject has dealt in particular with the clinical aspect. My intention is to deal very shortly with some pathological points. I think pathology has rendered very important service to medicine in putting on a sure basis the fact that general infection may commence by dissemination from the upper air-passages. English clinicians have done much to pave the way to this knowledge on the pathological side. The early experimental investigations in Germany demonstrated conclusively that a streptococcus might be present in the throat in diphtheria which would produce with some constancy a suppurative arthritis in animals. Dr. Paine and myself made another step when we showed that a

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diplococcus could be isolated from a rheumatic angina, which would on intravenous inoculation produce the lesions of rheumatic fever in the joints, heart, pleuræ and elsewhere, and which was present in those lesions in man. Then F. Myer and Menzer elaborated these early investigations and repeated them on a larger scale, so that now we know the stages in the process.

1. The micro-organisms get into the fibrous framework of the tonsils and thence reach the minute blood capillaries and lymphatics, so reaching the general circulation. In some cases the lymphatics appear to be the chief channel, and there is much enlargement of the cervical glands; in other cases the chief channel appears to be minute bloodvessels.

2. The micro-organisms have been isolated from the blood in man and found in the local lesions. We are, then, on firm ground—I do not say as regards the specific nature of any micro-organism, but as regards the fact under discussion, that a local focus in the upper air-passages may be the cause of a general infection.

There is another very important but disquieting fact that is established by pathology. It is that in many instances, when once the micro-organisms have gained access to the general system, they make for themselves new homes in the tissues. Few experiments are more convincing of this truth than the production of severe endocarditis in a rabbit by the diplococcus from rheumatic fever. A fortnight afterwards, if you cut a section of the valve, you may find literally thousands of virulent micro-organisms there. It is clear, therefore, that local treatment of the upper air-passages can only do a limited amount of good in infections of the rheumatic type, for each internal lesion is a local focus. But it holds good, as Dr. Hall has so well pointed out, that every effort should be made to arrest the process before it generalizes. I venture to think, nevertheless, that this great truth must be kept clearly in mind by those who are devoting their lives to a special study of the upper air-passages.

A third, an equally important point, is that at present we really do not know why it is that micro-organisms probably always present in the throat suddenly become dangerous. That borderline between pathogenic and non-pathogenic is, in my opinion, the weak spot of bacteriology at the present time. So far as rheumatism is concerned, it is, I think, very probable that there is a rapid multiplication of the micro-organisms in rheumatic angina. Such has been my experience and that of some German investigators, but this does not answer the question. Why? Because of the obscurity of this most pressing question, I have always fought

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hard against any assumption that whether a sore throat is septic or rheumatic or catarrhal is simply a question of the resistance of the patient. In opposition, I maintain that we do not know when we look at such a throat whether the fate of the patient turns upon his resisting powers or upon some special—specific, if you prefer that word—properties in the infective agent. Certainly, I think we shall admit, so far as diphtheria is concerned, much rests with the micro-organisms; and though in those other infections, so vaguely termed septic, we are at present in confusion, I hold firmly to the view that when you have great clinical differences, then you have some special factor in the infective agent. I admit the factor of personal resistance, but not as the explanation of the great differences that there are between the illnesses that follow rheumatic, septic, and catarrhal sore throats. In the search for these differences, difficult and slow though it may be, lies the hope of the discovery of the special poisons and their antidotes. In the massing together of these conditions as septic lies, in my opinion, the danger of a false confidence and imperfect methods of treatment.

There is one practical point which I have met with, and which, perhaps, is worth alluding to. On several occasions I have seen rheumatic fever directly follow tonsillotomy, though I do not say as cause and effect. When I say directly follow, I mean within a week of the operation, the patient being apparently in good health when the operation was done. So far as advising operation for rheumatic patients with diseased tonsils—and there are many such—all that can be said is, if their removal is for the general good of the patient, it is right; but it will not prevent further attacks, except in so far as improving the health protects against many infections.

The last point to which I should like to allude is this, that very probably infection from the throat may occur without any appreciable local symptoms. It is generally admitted that the most severe puerperal fever may occur from uterine infection with scarcely any local disturbance. The virulence of the infection is such that the unfortunate patient is in the position of an animal intravenously injected with micro-organisms. The same seems to occur in some cases of infection from the throat. This is a great practical difficulty, both in treatment and in getting an accurate idea of the frequency of infection from the upper air-passages. The local inflammation is only a measure of the local disease in the tonsil, and not of the tendency to general infection. My remarks, I am afraid, are somewhat limited in their scope, and have not the

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advantage of a special knowledge of the upper air-passages; but I should like to add that the subject under discussion appears to me a most important one, and I am much obliged to the President and Secretaries for giving me this opportunity of hearing and taking part in it.

Dr. Herbert Tilley limited his remarks to the part which the nose and naso-pharynx played as a source of general septic infection in two sets of circumstances:

1. When a chronic purulent focus exists the general health may suffer in a very profound and serious manner as a result of the constant absorption of the septic products from that focus.

2. When operative procedures have been carried out in these regions, very grave and even fatal results may ensue if due care has not been maintained to provide for and maintain surgical cleanliness of the wounds.

He said: With regard to the first point—viz., the effect upon the general health of continuous absorption of septic products from a chronic purulent focus—I would remind you of the loss of energy, lack of interest in the affairs of everyday life, general mental apathy, loss of appetite, wasting, and sallow, bespotted complexion, which are so often found in association with other local symptoms in connection with chronic empyema of the nasal accessory cavities. When such lesions have been present, I have been particularly struck by two facts which seem in almost direct opposition to one another.

In some patients—and this irrespective of sex—the evil effect of chronic absorption of septic products from such foci is very marked. None the less striking is the immediate improvement in the general health when the pus-producing focus has been efficiently treated. A patient who had suffered from suppuration of both maxillary antra for ten years, and who was in a miserable mental and constitutional condition when first seen, put on 10 pounds in weight during the six weeks which followed efficient drainage of those cavities; at the same time his tendency to melancholia and general depression entirely left him. Dr. William Hunter has drawn attention to the possible relation between such pus-producing centres and some of the graver forms of anæmia.

In the other class of patients the presence of a large bony cavity filled with the most fœtid pus of months or years duration seems to have no obvious effect upon their general health any more than it sometimes has upon their personal comfort or convenience. Such patients not unfrequently seek a consultation because they consider they are suffering from a little "post-nasal catarrh."

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They rarely complain of any digestive disturbances and loss of appetite, which may well account for many of the symptoms of the class first referred to. The question arises how far such a susceptibility may be due to lack of immunization, or how far is it due to deficiency in the secretion of free hydrochloric acid in the gastric juice, which, if poured out in sufficient quantity, would render inert any pus which had been swallowed. I am inclined to think the latter view the more probable one.

Passing now to general septic infection following operative procedures carried out upon the upper air-passages, I think we may congratulate ourselves that in spite of the great amount of operating which goes on, the instances of serious general septic infection are comparatively uncommon. Aside from surgical cleanliness on the part of the surgeon, this may be due to the free drainage which in most operations is naturally provided for, and to a peculiar capacity for healing which is possessed by the more vascular areas of the head and neck.

Accidents, however, have happened in the past, and will occur again in the future, if surgeons are careless, or even refuse to learn from their own failures and those of others. Let me give an illustration of what I mean. Some ten years ago I was suffering from a series of head colds to which I had long been subject. Between the acute attacks more or less nasal obstruction was always a trouble, and necessitated constant mouth-breathing. I consulted a friend, who cauterized the mucous membrane of my left inferior turbinal. No antiseptic dressing or application was applied to the eschar, and with his consent I followed my daily routine of work. The same evening I felt very unwell, had a slight rigor with intense throbbing of the left side of the face and of the left tonsil. For the next three days my temperature varied between 101° and 103° F., and on the fifth day an acute abscess of the left tonsil discharged itself, after which convalescence set in.

Last November I saw in consultation a lad who four days previously had had a similar operation performed in Paris. He was allowed to travel to England the same day. Towards evening he felt unwell, with some pain in the right side of the face and a sore throat, which was also located to the same side. He arrived at Ealing during the next day feeling very ill, with intense sore throat and swelling of the glands under the angle of the jaw; temperature 101·6° F. When I saw him on the fifth day he had a very inflamed throat, more especially the right side; swallowing was impossible except for small quantities of liquids; the tongue thickly furred; temperature 103° F. There was a very great

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swelling of glands under the right angle of the jaw; in fact, the whole side of the neck, from the tip of the mastoid to the clavicle, was a red brawny mass acutely tender on pressure and semi-fluctuating in parts. The patient was altogether very ill. Incisions were made into the swelling, drainage-tubes inserted, constant fomentations applied, antistreptococcus serum injected, and after a very anxious illness and tedious convalescence the patient was restored to health. It was a case of streptococcus infection.

In both cases the history would seem to show that infection almost certainly entered by the wound of the nasal mucous membrane, and had the eschar in either case been carefully dressed, it is more than probable that no harm would have resulted. Hence before cauterizing the nasal mucosa the parts should be thoroughly cleansed with a simple alkaloid wash, and after the cauterization some glycerine of carbolic acid should be rubbed into the eschar by means of a cotton-wool mop.

From want of similar care I have known the greater part of the cartilaginous nasal septum slough away after Asch's operation on the septum, the patient suffering in the meantime from constitutional symptoms of septic poisoning.

After the radical operations practised for the cure of chronic empyema of the maxillary antrum, it is not usual for grave constitutional symptoms of septic poisoning to arise; but on one occasion I have known intense cellulitis followed by abscess in the soft tissue of the cheek. It is not uncommon for the temperature to rise for a degree or two during the three days following the radical operation, especially if the sinus be packed tightly with gauze. In such cases the soft tissues of the cheek are often swollen and painful. To avoid these complications, I never pack the antra at all after the operation, but keep them clean by frequent irrigations during the ten days or so after the operation.

With regard to the radical operation upon the frontal sinus as it is practised by most of us to-day (viz., removal of the greater part of the anterior wall, coupled with enlargement of the frontal nasal canal and removal of the neighbouring diseased anterior ethmoidal cells, which is followed by gradual and complete obliteration of the sinus), it may be said to be free from risk to life, provided surgical cleanliness be maintained and the surgeon has a practical working knowledge of the anatomy of the parts concerned. But this comparative freedom from risk has been only painfully attained by those who have spent much time, much anxiety and much thought in the evolution of the operation as it is practised to-day. Many patients have succumbed to general septic infection

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following, and due to, the operation carried out for the relief of chronic empyema of the frontal sinus. Fifteen such cases are known to me, two of them in my own, and, I am glad to say, my earlier, practice. In every case the fatal result has arisen from a spreading septic osteomyelitis of the frontal bone resulting from imperfect drainage of the bony cavity following the radical operation. To avoid such disaster, let me recommend you to make a large opening into the nose, and never completely sew up the external wound nor pack it very tightly with gauze.

Again and again has this warning been given in our Societies and our journals, and yet it would seem necessary to repeat it, for only a very short while ago a case which I had seen in consultation died within five weeks of the radical operation from general septic infection arising from neglect of the very points which I have mentioned.

The sphenoidal sinuses have recently been receiving a consideral amount of surgical attention. Not infrequently they are affected simultaneously with empyema of the neighbouring accessory cavities, and most surgeons are content to remove the middle turbinate bone and the front wall of the sphenoidal sinus so as to insure free drainage. If this be followed by free irrigation of the sinus with suitable antiseptics great relief will ensue. Anything like thorough curettage of the sinus walls is extremely dangerous, for it is absolutely impossible to see weak spots or detect exposed dura mater. I was not at all surprised, therefore, to hear that a surgeon who curetted a sphenoidal sinus some three weeks or a month ago lost his patient through septic meningitis two or three days after the operation.

With regard to systemic infection following operations upon the post-nasal space, and again confining myself to septic infection, I presume we have all been unfortunate enough to have had one or two cases in which after the operation for adenoids the patient has had a high temperature for a week or so, associated with enlarged and tender glands in the neck and constitutional symptoms indicating a general septic infection. Two consecutive cases have occurred in my practice, in one of which no sponge was used during the operation, and the question arose as to whether the modified Gottstein curette was at fault. I believe so, for I found the blade was completely cracked through, and probably harboured some septic organisms. I procured a new instrument, and have had no further trouble. Septic infection followed by death after the operation for adenoids is not unknown; in one case it was proved to be due to escape of sewer-gas into the patient's room.

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The following is an interesting case of general septic infection following upon an operation for the removal of a pre-existing focus of suppuration in the naso-pharynx:

For twelve months a lady had suffered from periodical attacks of severe pain limited to the left side of the head, and which seemed to radiate from behind the left angle of the jaw, where pain was most intense. On one or two occasions there had been a discharge of pus from the left ear. After three or four days of suffering she "felt something burst," and experienced an unpleasant taste and smell, while the symptoms immediately disappeared. On examining the post-nasal space with the finger, I burst an abscess which was located in the remains of an old adenoid growth. The odour from it was extremely fœtid. An attack of septic sore throat immediately supervened, the right maxillary antrum became infected, and after two days of great facial pain the sinus discharged pus very freely. Some ten days afterwards, when the patient was fairly strong, I removed a large tough adenoid growth in which the abscess with thickened walls was situated. The right antrum was also drained through the alveolus. At the close of the operation I swabbed out the post-nasal space with chloride of zinc solution (grs. 40, ad ℥j), but in spite of this she again suffered from a sort of septic throat, inflamed glands in the neck, and her temperature varied from 100° to 102° F. for the following week. An interesting feature in this remarkable case was the appearance of a scarlet punctiform rash on the third day after the operation, which appeared on the neck and upper part of the chest and back. From here it spread over the face, the lower chest, the abdomen, to the extensor surfaces of the arms and thighs, then to the flexor surface of limbs. The tissues of the eyelids were swollen and deep red to crimson. The rash itched intensely, lasted six days, and desquamated slightly on the face and neck. The tongue was coated in the middle and was red at the edges; there was no prominence of papillæ; no albuminuria. The rash at first sight reminded one very forcibly of scarlet fever, but the history of the disease and the general course of the symptoms led us to regard the eruption as a "septic rash."

The case affords an excellent illustration of a chronic localized suppuration giving rise to both local and general symptoms of an uncommon nature, and shows that in spite of strong antiseptic precautions a still more acute general and local infection may follow attempts at removal of the original source of trouble.

Dr. D. R. Paterson (Cardiff) spoke of cases of nephritis occurring

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in patients the subjects of atrophic rhinitis, which he considered had a causal relationship. He also cited cases of tuberculosis developing in subjects of ozæna. He regarded the supra-tonsillar fossa as a vulnerable spot and a starting-point for infectious processes.

Dr. R. H. Woods (Dublin) regretted that the mouth had not been brought within the scope of the discussion, as septic conditions in the buccal cavity were frequently the cause of enlarged cervical glands. He gave details of a case of primary nasal tuberculosis which was followed by tuberculous infection of the larynx, the lungs remaining normal. He had observed a case of Hodgkin's disease in which infection had undoubtedly occurred from the tonsil.

Dr. Logan Turner referred to obscure septic conditions arising without any obvious cause in which infection probably occurred in the upper respiratory tract.

Dr. W. Lamb (Birmingham) described a case of acute laryngitis with œdema of the glottis, and which, after a week, ended fatally through double pneumonia; the laryngeal symptoms had subsided when the pneumonia developed. He also said that the site in the larynx referred to by Dr. Jobson Horne as the "vulnerable spot" had been indicated as a site of infection in typhoid fever.

The President (Dr. Watson Williams), in reviewing the discussion, emphasized the importance of hypertrophy of the tonsil as a cause of secondary anæmia through absorption of toxic products from the multitude of septic and saprophytic micro-organisms in the crypts.

Replies,

Dr. de Havilland Hall, in reply, remarked that Dr. Poynton had not, he thought, quite understood him. He said that rheumatism ran the course of an attenuated pyæmia. By this he did not intend to convey that rheumatism was of pyæmic origin, but that in its symptoms it resembled pyæmia; indeed, as Hilton Fagge had pointed out, the differential diagnosis between the two diseases was sometimes quite impossible. Dr. Tilley's remarks on the necessity of strict antiseptic precautions in the employment of surgical procedures in the upper respiratory tract should receive universal attention, and lead to a diminution of the lamentable cases of septic infection which had resulted from want of care. He had no experience of the connection between chronic atrophic rhinitis and renal disease, but it might possibly be the explanation of some cases in which the etiology had not yet been demonstrated. As regarded Dr. Paterson's suggestion that atrophic rhinitis led to

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tuberculosis, he would rather look upon the atrophic rhinitis and the tuberculosis as being due to a common vulnerability of the mucous membrane. He agreed with Mr. Woods that it would not do that it should go forth to the world that this section regarded the mouth as part of the respiratory tract. If it had been included, he would have mentioned a case of acute septicæmia ending fatally resulting from carious teeth. As a pendant to Mr. Woods' case of lymphadenoma, he mentioned a case of sarcoma of the left tonsil, which was followed by diffuse sarcomatosis.

Dr. Jobson Horne, in his reply, expressed gratification at the discussion having brought out so much of interest. He thought that Dr. Poynton's paper rather added to the importance of local treatment in all cases of tonsillitis in view of the danger of systemic infections. As regarded pulmonary tuberculosis developing in the subjects of atrophic rhinitis with ozæna, he was inclined to take the same views as Dr. Paterson in holding the nasal condition to be a causative factor and not a mere coincidence. Dr. Horne was glad to learn that the site in the larynx he had drawn attention to as a vulnerable point in certain infective diseases—endocarditis, Hodgkin's disease, tuberculous lymphadenitis, lympho-sarcoma—had already been regarded in a similar light in typhoid fever.

SOCIETIES' PROCEEDINGS.

NINTH ANNUAL MEETING OF THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL, AND OTOLOGICAL SOCIETY.

Held in Lexington, Ky., April 30, *May* 1 *and* 2, 1903.

President: J. A. Stucky, M.D., of Lexington, Ky.

(*Continued from p.* 555.)

Dr. Edward B. Dench, of New York, read a paper on *The Technique of the Radical Operation for Otitis Media Suppurativa Chronica.*

He said that it was imperative to shave the head for 3 inches around the ear. Women objected strongly to this, but it should be done, nevertheless. The field of operation should be sterilized in the ordinary way, and an antiseptic dressing applied until the time of operation. The initial incision began ¼ inch behind the

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